insipidus includes several conditions: (1) True diabetes in which, for some reason, sugar is temporarily absent. There may or may not be concomitant azoturia. Usually, the sugar and the nitrogen fall *pari passu*. (2) Unrecognized interstitial nephritis. (3) Cardiac and other complications causing increase of arterial pressure and consequent diuresis. (4) Excessive ingestion of salts and sugar, beer, etc., which lead to the absorption into the blood of unusual quantities of water; the sugar, especially, largely disappears, so that no more than the normal or a slightly exaggerated trace is found in the urine. (5) Excessive imbibition of water as such or in various beverages. (6) Central nervous or ganglionic disturbances which exalt the renal function.

It is obvious that the foregoing classification is inexact and that the different groups overlap. Only the last really deserves to be termed an essential polyuria or diabetes insipidus, and its explanation is at present far from satisfactory.

Perversions of the urinary secretion or, rather, excretion cannot be considered in detail. All things considered, the urinary examination is of more value to us than that of the stomach contents or, *a fortiori*, of the feces. The test for indican deserves special mention, as it is within the power of any practitioner, is sadly neglected, and is often the key to the whole situation.

HEART-BLOCK (ADAMS-STOKES DISEASE).

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The clinical syndrome known as Adams-Stokes disease has been recognized since its first description by Adams in 1827. It is only within recent years, however, that an increased knowledge of cardiac physiology and pathology has enabled an intelligent conception of its cause and a reasonably sufficing explanation of its symptoms. The subject has recently been discussed in detail by Ashton, Norris, and Lavenson.[1]

The facts and theories, to which they direct attention, serve to explain very exactly the manifestations occurring in the Adams-Stokes syndrome. The characteristic symptoms are: (1) Slow arterial pulse, 30, 20, or less per minute; (2) brief syncopal, apoplectiform, or epileptiform attacks; and (3) pulsation of the veins in the neck, these pulsations occurring at intervals more frequent than the radial pulse-beat, the ratio being from 2 to 1 to 4 to 1 or more. The parallelism between the phenomena resulting from experimental constriction of the auriculoventricular bundle and those occurring in Adams-Stokes disease is so close that it is quite safe to attribute the latter to an interruption of the normal communication from auricles to ventricles either by functional weakness or organic defect.

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[1] Amer. Jour. Med. Sci., 1907, cxxxiii, 28.

The case which I wish to report is that of a man, forty-three years of age. He was seen by me on two occasions through the courtesy of his physician, Dr. Joseph G. Leiter, of Brooklyn. The patient, a business man, had always been extremely temperate and regular in his mode of life, with no bad habits in eating or drinking. The family history presented nothing of interest. His personal history told of typhoid fever twenty years, and lobar pneumonia two years previous to his last illness. Since the typhoid fever, and perhaps before it, his pulse had always been slow. During his pneumonia, Dr. Leiter never found it over 42 per minute. His final illness, which began ten days before his death, was ushered in by fever (not higher than 101°), and pains in the larger joints. The temperature and joint pains rapidly subsided, but on the third, fourth, and fifth days he had a brief epileptiform convulsion, and the radial pulse went down to 28. Subsequently the convulsion seizures occurred more frequently until they became almost continuous in the last day or two; the pulse slowed down still more, varying from 14 to 20 per minute between the attacks. The seizures began with a marked pallor, the pulse dropping to 6 beats or less per minute, nearly twenty seconds elapsing between successive beats on one occasion. The eyes rolled up and to the right, the pupils dilated, the head turned to the right, the lips were cyanosed, and the arms became rigid and underwent slight clonic movements. The unconsciousness varied from partial to complete and was always brief. Just previous to the end of the seizure the pulse began to beat a little more rapidly and much more strongly, the face flushed, and consciousness returned. Vomiting occurred with some frequency before the attacks. Finally the patient passed quietly away.

During my two examinations, which were made during the last three days of the illness, the auricular pulsations in the veins of the neck were plainly visible. They varied from 80 to 130 per minute, and the auriculoventricular pulse-ratio was suggestive, not of a partial, but of a complete heart-block, the ventricular systoles bearing no definite relation to those of the auricles. In contradistinction to Stengel's experience, it was possible in this case to hear (with a Bowles stethoscope) faint fluttering sounds over the third interspace to the left of the sternum, which sounds were distinctly synchronous with the visible auricular pulsations.

The autopsy, made by Dr. Raymond Clark, assistant pathologist to the Seney Hospital, showed, in synopsis, the following findings: Hypertrophy and dilatation of the left auricle and ventricle and right auricle, and hypertrophy of the right ventricle; moderate mitral insufficiency; thickening at the base of the mitral cusps; and an increase in the amount of adipose tissue in the pericardium over the right ventricle at the junction of the auricles and ventricles and along the coronary arteries. Microscopically there was fatty infiltration and degeneration in the walls of the right ventricle, and, to a less extent, in those of the left ventricles; and no pathological changes in the coronary arteries. The kidneys showed an acute exudative nephritis with fatty changes; the liver a slight interstitial nephritis, with fatty degeneration of the liver cells, and a moderate venous congestion.

Careful sectioning of the bundle of His, compared with similar sections from a normal heart, showed that the bundle was infiltrated with fat, and had undergone fatty degeneration to such an extent that it was atrophied to not more than one-fifth of its normal size. The result of the autopsy in this case, therefore, tends to confirm the later theories in regard to the nature and causation of Adams-Stokes disease.

POSITION OF THE MOTOR AREAS OF THE HUMAN CORTEX.

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Case I.—*A rapidly growing glioma of the right motor area (precental convolution) occupying the leg centre, the centres for the arm and face being destroyed by softening. Clinically focal Jacksonian attacks, usually beginning in the left leg, and not preceded by a sensory aura, the leg becoming totally, the arm and lower facial muscles partially, paralyzed as to motion but not as to sensation.*

Mrs. M., aged seventy-six years, was seen on August 10, 1900. Her father died of cancer of the stomach, her mother of pneumonia. She lost two brothers—one of pneumonia, the other of la grippe. Three sisters are living and well. There is no history of insanity or nervous diseases in the family.

The present illness began ten weeks ago with a spasm of the whole left side. This spasm began as a jerky motion of the leg, which was followed by similar movements of the arm and face. The spasm was preceded by no signal symptoms, such as numbness, tingling, or formication, nor by headache, nausea, or vertigo. These attacks, numbering from three to twenty daily, have been repeated ever since. They always begin in the leg or foot, extend upward to the thigh and then involve the arm and face. They never have been followed by loss of consciousness. Since the first unilateral convulsion, she has been slightly paralyzed on the whole left side. She never experiences numbness or pain in the extremities. She states that